Science & Solutions



What's Wrong With My Birds?

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The What's Wrong with My Birds? series offers practical 1-page articles on common issues in modern poultry production. Each differential diagnosis identifies potential cause, description of problem, checklist and corrective actions to help you to maintain production performance.







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What's Wrong with My Birds? Part 1: Oral lesions



Science & Solutions presents a handy checklist for diagnosing poultry mycotoxicosis. Cut this out and take it along with you to the farm!

Diagnosing common poultry ailments correctly and precisely can be a challenge even for experienced vets, nutritionists or farm managers. Differential diagnosis is especially difficult in the case of mycotoxin-related problems as symptoms vary greatly and may be further complicated by the synergistic effects caused by the co-occurrence of more than one type of mycotoxin in the feed.



				ANAGEN
_	Potential cause	Description of problem	Checklist	Corrective actions
MYCOTOXINS	T-2 toxin (T-2) Diacetoxy- scirpenol (DAS)	T-2 and DAS have a dermatotoxic action, thus causing lesions to the epithelium, increasing the speed of epithelial cell renovation.	 □ Positive for T-2 and/or DAS in raw materials (ELISA) or feed (HPLC) □ Origin of raw materials from supplier/ region with history of T-2/DAS contamination □ Histopathology: Proliferating epithelial cells and hepatic vacuolization □ Overall decrease in flock performance 	 □ Check average contamination levels □ Use Mycofix® at a correct dosage level □ Avoid feed bins or feed/water lines that have become contaminated by stale, wet or moldy feed
NUTRITION	Feed granulometry	Small particles of feed block saliva ducts, which may result in oral lesions.	 □ Pelletized feed: Fine particles >20% □ Mashed feed: Check mean particle diameter □ Histopathology: Presence of inflammatory cells and bacteria □ No overall decline in flock performance 	☐ Adjust the pelleting process ☐ Increase the sieve diameter ☐ Use pellet binders to improve pellet quality
MANAGEMENT	Liquid methionine	Methionine dripping in the application system produces points of high methionine concentration in the feed.	 □ Methionine injector dripping inside masher □ Histopathology: Infiltration of inflammatory cells and necrotic lesions □ No overall decline in flock performance 	☐ Clean/replace methionine injectors
Σ	Organic acids	Excessively high concentrations of organic acids in the feed lead to caustic lesions in the oral mucosa.	 ☐ Acids injector dripping inside masher ☐ Histopathology: Infiltration of inflammatory cells and necrotic lesions ☐ No overall decline in flock performance 	☐ Clean/replace acid injectors ☐ Adjust dosage of organic acids
ı	High temperatures	More frequent drinking during hot periods increases feed residues in beaks.	 ☐ Histopathology: Infiltration of inflammatory cells and necrotic lesions ☐ Possible decline in flock performance ☐ Increased mortality 	☐ Apply vitamins in water ☐ Apply organic acids in water ☐ Increase chlorine level in water
	Copper sulphate	Concentrations between 0.05 to 0.2% in feed and drinking water can promote oral lesions.	 □ Check concentration of CuSO₄ in premix □ Check concentration of CuSO₄ in water □ Check if water dosing system is working correctly (if applicable) 	☐ Apply group B vitamins and K ₃ vitamin in water ☐ Correct set-up of the water dosing system
PATHOGENS	Candida albicans (Candidiasis)	The yeast <i>C. albicans</i> can lead to lesions in the crop that can extend to other parts, including the mouth. More common in birds with longer lifespans, such as layers and breeders.	☐ Histopathology: Fungal hyphae present in affected mucosa	☐ Nystatin or diflucan or imidazoles such as ketoconazole, fluconazole, etc. as treatment
	Fowl pox (Avian pox)	Viral disease caused by Poxviridae (Avipoxvirus) often leads to cutaneous lesions on head, neck, legs and feet. • Dry pox: Raised, wart-like lesions on feathered areas (head, legs, vent, etc.) which heal in about 2 weeks. • Wet pox: Canker-like lesions in the mouth, pharynx, larynx, and trachea.	 □ Flock history and presence of typical lesions □ Laboratory diagnosis by tissue or transmission studies 	 ☐ Use preventive vaccination depending on prevalence and season (typically fall) ☐ Treat affected birds with antibiotics to reduce secondary infection, although the disease has to run its course
	Protozoans	Protozoans are more prevalent in birds with a longer lifespan, such as layers, breeders and turkeys, game birds and/ or free-range birds.	☐ Histopathology: Microscopic examination of a smear of mucus or fluid from the throat demonstrates the presence of trichomonads	☐ Separate chronically infected birds from breeding birds
	Trichomonas gallinae	First lesions appear as small, yellowish areas on the oral mucosa.	☐ Cankers, also known as "yellow buttons" — yellow, rounded areas with central caseous necrotic foci	☐ Use nitroimidazoles (not approved in US by FDA and prohibited in the EU)
	Histomonas meleagridis	Also known as histomoniasis or blackhead disease. Common in commercial turkeys and chickens.	☐ Cecal inflammation, ulceration, thickening of wall, ceca containing yellowish cheese-like exudate	☐ Use nitroimidazoles (not approved in US by FDA and prohibited in the EU)

What's Wrong with My Birds? Part 2: Gizzard lesions



Science & Solutions presents a handy checklist for diagnosing poultry mycotoxicosis. Cut this out and take it along with you to the farm!



Diagnosing common poultry ailments correctly and precisely can be a challenge even for experienced vets, nutritionists or farm managers. In the case of mycotoxin-related problems, differential diagnosis can be especially difficult as symptoms vary greatly. The following table provides an overview of the potential causes and a checklist of corrective actions; however, please exercise due caution and discretion in use.

	Potential cause	Description of problem	Checklist	Corrective actions
MYCOTOXINS	Cyclopiazonic acid (CPA) Deoxynivalenol (DON) and/or T-2 toxin (T-2)	Lesions develop in the proventriculus, gizzard, liver and spleen. The proventriculus is dilated and the mucosa is thickened and sometimes ulcerated.	☐ Positive for CPA, DON and/or T2 in raw materials (ELISA) or feed (HPLC) ☐ Raw materials originating from supplier/ region with history of CPA contamination ☐ Histopathology: Proventriculus hyperplasia of mucosa with heavy infiltration of lymphocytes ☐ Overall decline in flock performance	 □ Check average contamination levels □ Use Mycofix® at a correct dosage level □ Avoid feed bins or feed/ water lines that have become contaminated by stale, wet or moldy feed
AGEMENT	Copper sulphate	${\rm CuSO_4}$ can promote gastric lesions especially at the gizzard level.	☐ Concentration of CuSO ₄ in premix ☐ Concentration of CuSO ₄ in water ☐ Water dosing system is working properly (if applicable)	☐ Apply group B vitamins and K ₃ vitamin to the water ☐ Correctly set-up the water dosing system
MAR	Acetylsalicylic acid and sodi- um salicylate	Use of salicylates may induce proventriculus and gizzard ulceration.	 □ Dosage of salicylates used (check overestimation of feed intake in feed restricted animals) □ Mixability of commercial product in water 	 □ Avoid low quality products (low mixability, low homogeneity in water) □ Adjust the feed intake of feedrestricted animals
NUTRITION	Biogenic amines (Gizzerosine)	Low quality/ over-processed fishmeal can result in high levels of gizzerosine. Hyper-production of HCI in the proventriculus causes erosions in the gizzard.	☐ Level of gizzerosine in raw materials (especially fishmeal)	☐ Lower the level of fishmeal in diets ☐ Avoid using low quality fishmeal ☐ Replace standard fishmeal with low temperature (LT) fishmeal
ı	Rancid fats	Low quality fats (long storage, overheating) can contain high levels of superoxide radicals and hydroxyl radicals.	☐ Quality of fats in term of peroxide value, rancidity and free fatty acids	☐ Avoid low quality fats ☐ Use low quality fats in the grower/ finisher phases ☐ Replace animal fats with vegetable fats
ı	Tannins	Toxic levels of tannins in the feed cause oesophageal and gastric edema, hemorrhagic ulceration, necrosis and sloughing of the mucosal lining.	☐ Level of tannins in some raw materials (sorghum) and in tannin-based products	 ☐ Use high quality tannin-based product (chestnut is preferred to quebracho) ☐ Reduce % of sorghum in high-tannin diets
PATHOGENS	Adenovirus serotype 1	Vertically transmitted, usually subclinical but provides more exposure to secondary bacterial infection. • Group I is exhibited through inclusion body hepatitis (sudden onset of mortality, typically 10% and rarely up to 30%) or hydro pericardium (same symptoms as IBH, but severe mortality at 20-80%). • Group II is exhibited through hemorrhagic enteritis and marble spleen disease in turkeys, and avian adenovirus group II splenomegaly in chickens. • Group III affects most poultry due to the egg drop syndrome.	□ Isolation of serotype I, II or III from the lesions by serological assays	☐ Use inactivated vaccines (only available for group 1) ☐ Check the breeding stock and eliminate affected birds
	Infectious bursal disease (IBDV/ Gumboro)	IBDV is very immunosuppressive and causes lesions at the junction of the proventriculus-gizzard.	☐ Maternal antibody titers are very low in day-old chicks	 ☐ Implement/ correct vaccination program in breeders ☐ Change from mild- to strong-reaction vaccine ☐ Correct vaccination age (Deventer formula) ☐ Increase biosecurity level

What's Wrong with My Birds? Part 3: Fatty Liver



A handy checklist for diagnosing poultry mycotoxicosis that you can cut out and keep with you for reference.

Diagnosing common poultry ailments correctly and precisely can be a challenge even for experienced vets, nutritionists or farm managers. In the case of mycotoxin-related problems, differential diagnosis can be especially difficult as symptoms vary greatly. The following table provides an overview of the potential causes and a checklist of corrective actions; however, please exercise due caution and discretion in use.



	Potential cause	Description of problem	Checklist	Corrective actions
NUTRITION	Energy-protein ratio	Excessive energy in diets can cause lipidosis and fatty liver problems.	☐ Carbohydrate level in diet☐ Energy-protein ratio in diet☐	 □ Avoid high carbohydrate diets, especially in summer □ Adopt proper energy-protein ratio □ Apply amino acids in drinking water
	Rancid fats	Peroxides can impair liver activity, creating fatty liver.	□ Quality of fats in term of: peroxide value, rancidity and free fatty acids	 □ Avoid low quality fats □ Use low quality fats in the grower/ finisher phases □ Replace animal fats with vegetable fats □ Apply choline chloride and Vitamin B in feed or water
MYCOTOXINS	Aflatoxins (Afla)	Young animals: fibrosis of liver leads to hardening of the organ. Older animals: hepatic lipidosis, with softening of the organ.	 □ Positive for Afla in raw materials (ELISA) or feed (HPLC) □ Raw materials originating from supplier/region with history of aflatoxin contamination □ Histopathology: Check other target organs of Afla (ex. Liver) □ Overall decline in flock performance 	 □ Check average contamination levels □ Use Mycofix® at a correct dosage level □ Avoid feed bins or feed/water lines that have become contaminated by stale, wet or moldy feed
MANAGEMENT	Hormone status	An over-stimulation of egg production may lead to excessive levels of estrogen in the blood that facilitate fat storage in the liver and the occurrence of fatty liver, especially in layers and breeders.	□ Management of laying birds	☐ Improve management of laying birds ☐ Correct lighting program
PATHOGENS	Viral hepatitis (IBH – viral inclusion body hepatitis)	Adenovirus causes yellow/hemorrhagic liver and focal necrosis. Symptoms include immunosuppression, diarrhea, anorexia, depression, ruffled feathers, especially in the region of head and neck. Group I is exhibited through inclusion body hepatitis (sudden onset of mortality, typically 10% and rarely up to 30%) or hydro pericardium (same symptoms as IBH, but severe mortality from 20-80%). Usually occurs in chickens older than 3 weeks.	 □ Clinical signs only several hours prior to death: pale comb and wattles, depression and apathy □ Up to 30% mortality □ Necropsy: Macroscopic lesion is the enlarged, dystrophic liver with yellowish color and crumbly texture and enlarged kidneys □ Histopathology: Detection of intranuclear inclusion bodies □ Isolation of serotype I or II or III from the lesions by serological assays 	 ☐ Use inactivated vaccines (exist only for group I) ☐ Check the breeding stock and eliminate the affected birds

For more information, visit www.mycotoxins.info

DISCLAIMER: This table contains general advice on poultry-related matters which most commonly affect poultry and may be related to the presence of mycotoxins in feed. Poultry diseases and problems include, but are not confined to the ones present in the table. BIOMIN accepts no responsibility or liability whatsoever arising from or in any way connected with the use of this table or its content. Before acting on the basis of the contents of this table, advice should be obtained directly from your veterinarian.



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What's Wrong with My Birds? Part 4: Impaired feathering/feather loss



In poultry, feathers serve important roles in terms of protection and insulation of the body. While moulting, or renovation of older feathers by new ones, is a natural process occurring in mature layers upon completion of a laying cycle (which itself can be influenced by many factors), feather loss or impaired feathering may be indicative of other problems in the farm.

Feather-related problems in poultry can be roughly divided into two groups, either:

- They are not properly developed (linked to feather formation) which is often related to nutrition or the presence of mycotoxins.
- They are pulled off by birds (feather pecking), which is a management-related issue

In each case it is critical to understand the foundation of the problem so that it can be properly solved (see table right).

Stressful conditions in the barn, especially during brooding, such as heat, cold and existence of air currents, amongst others, can result in feather loss and poor feather quality in the birds. In this case, it is crucial that the behavior and interaction of animals is observed. Often, feather pecking and pulling can also be triggered by inadequate intake of nutrients. Due to the high protein content in feathers, higher protein levels in feed may encourage faster feather development and shedding.

Imbalance of amino acids in the feed, particularly sulphur amino acids cysteine and methionine, may cause feather abnormalities and/or rough feather appearance.

The dermotoxic effect of trichothecene mycotoxins, such as T-2 toxin and others, may also contribute to low feather quality along with other negative effects, such as oral lesions and decreased performance.

Overall, excessive feather loss or impaired feathering adversely affects feed conversion as birds have to allocate extra energy from the diet to compensate for heat loss.

As such, management, housing and nutrition should be optimized to reduce this occurrence. In terms of mycotoxins, prevention can be undertaken through the use of a proper mycotoxin risk management tool which adsorbs and/or biotransforms mycotoxins, thus eliminating their toxic effects for the animals, while guaranteeing liver and immune protection.

Checklist	Corrective action
Potential cause: MANAGEMENT: Temperature of	barn
Temperature of barnHumidity of barnVentilation system	 Improve management of barn Correct temperature, ventilation rate and humidity according to management manuals
Potential cause: MYCOTOXINS: T-2 toxin (T-2)/Do Other trichothecenes	eoxynivalenol (DON)/
 Positive for trichothecenes in raw materials (ELISA) or feed (HPLC) Raw materials originating from supplier/ region with a history of trichothecenes contamination Histopathology: Check other target organs for trichothecenes (ex. liver, for hepatic vacuolisation) Decline in overall flock performance Potential cause: NUTRITION: Amino acid (AA) december of the patic vacuolisation of the patic vacuolisation of the performance Potential cause: NUTRITION: Amino acid (AA) december of the patic vacuolisation of the performance Level of Total Sulphur Amino Acids (TSAA) in diet Ratio TSAA/Lys/Arg/Thr AA scale at feed mill 	 Check the average contamination levels Use Mycofix® at the correct dosage level Avoid contamination of feed bins or feed/water lines by stale, wet or mouldy feed efficiency/unbalance Increase level of synthetic Amino Acids (AA) in low digestible diets (high levels of by-products)
Potential cause: MANAGEMENT: Red mites	
Presence of red mites in the barn during the night.	 Flame cages during withdrawal period Clean egg belts during withdrawal period. Increase biosecurity level

References are available on request

Use plastic egg belts whenever

possible

For more information, visit www.mycotoxins.info

but may be important to consider.

Note: Pathogens were excluded from the table due to space constraints

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What's Wrong with My Birds? Part 5: Egg production/quality problems





Good, stable egg production and good quality are of utmost importance. Bad management practices, feed and environment-related issues and diseases are some of the factors which may negatively impact egg production and egg quality.

Also animal-related factors, such as age and strain of layer birds, must not be disregarded. Older birds and birds after molting are known to produce bigger eggs with thinner shells and indigenous strains cannot quite compete with commercial layers in terms of number of eggs produced.

Several management issues (see table) may lead to nervous birds and/or traumatic lesions in the ovary which cause poor egg quality (fragile shell/bloodspots/meat spots). Nutrition-wise, improper balance of calcium, phosphorus and vitamin D may lead to thin egg shells. Also large amounts of Lucerne/ alfalfa meal in diet can lead to blood spots caused by vitamin K antagonists in this feed ingredient. Interestingly, the use of the drug sulphaquinoxaline may have the same effect as mineral imbalance. In terms of pathogens, Infectious Bronchitis (IB) causes respiratory disease and kidney damage in growers and oviduct infection in adult hens which can cause wrinkled egg shells as well as a reduction in eggs laid.

Due to the liver and kidney toxicity mycotoxins may negatively impact egg and shell formation, leading to poor egg and shell quality (pale eggs/small, fragile shell/bloodspots/meat spots). For mycotoxin-related problems, prevention can be undertaken through the use of a proper mycotoxin risk management tool which adsorbs and/or biotransforms mycotoxins, thus eliminating their toxic effects for the animals, while guaranteeing liver and immune protection. The Mycofix® product line from BIOMIN combines the three strategies – adsorption, biotransformation and bioprotection which work together to prevent the hazardous effects of mycotoxins in poultry flocks.

Checklist Corrective action

Potential cause: MANAGEMENT: Nervous birds/traumatic lesions in the ovary

- Lighting program
- Temperature of the barn
- Presence of frights and disturbances in the barn that may get birds nervous
- Correct lighting program
- Correct temperature of the barn
- Improve management of laying birds

Potential cause: MYCOTOXINS: Aflatoxins (Afla), Cyclopiazonic acid (CPA), T-2 toxin (T-2), Ochratoxin A (OTA)

- Positive for Afla, CPA, T-2 and/or OTA in raw materials (ELISA) or feed (HPLC)
- Raw materials originating from supplier/ region with history of mycotoxin contamination
- Histopathology: Check other target organs
- Decline in overall flock performance
- Check average contamination levels
- Use Mycofix® at the correct dosage level
- Avoid feed bins or feed/water lines to become contaminated by stale, wet or mouldy feed of these mycotoxins (for example kidneys, liver)

Potential cause: NUTRITION: Mineral/vitamin imbalance

- Calcium/phosphorus balance in diets
- Calcium carbonate particle size
- Correct mineral and vitamin balance
- Correct calcium carbonate particle size

Potential cause: NUTRITION: Vitamin K antagonists

- Content of lucerne/alfalfa meal in diets
- Presence of sulphaquinoxaline in diets
- Correct amount of lucerne/alfalfa meal in diets
- Correct medication program

Potential cause: PATHOGENS: Infectious bronchitis (IB)

- Laboratory tests to confirm the presence of the coronavirus in a swab or tissue sample
- Vaccination program must be adapted to the demands of the field situation in each particular area/epidemiology

Potential cause: GENETICS: Bird strain

- Check with genetic supplies (some strains more susceptible to blood spots)
- Replace genetics if necessary

References are available on request

For more information, visit www.mycotoxins.info

What's Wrong with My Birds? Part 6: Carcass bruising



The consumer's decision making process when purchasing poultry products mainly takes into account appearance, hygiene and flavor. To help guarantee that the best quality product reaches consumers, several procedures should be in place.

Veterinary inspection at the time of slaughter aims to guarantee that poultry carcasses are free from disease or fecal contamination. In the presence of one (or both) contaminations, carcasses are condemned and withdrawn from the food chain.

Carcass bruising/hemorrhaging is one of several reasons leading to carcass downgrading (reduced quality) or condemnation in the slaughterhouse. It is caused by the breakage of blood vessels and subsequent leakage of blood into tissues without skin rupture.

It is difficult to determine whether they occur at the farm, during transport or at the plant; therefore, any major financial losses that result are usually absorbed by the slaughterhouse.

According to scientific literature, the colour of the bruise may be indicative of the age of the injury with red to dark red being recent bruises (\leq 12 hours) and light green, yellow-orange and yellow ones being older (\geq 24 hours).

Some 90% of bruising occurs within 12-24 hours before processing, with breast, wings and legs the most frequently affected parts. The potential causes are inadequate flock density in the grow-out house and/or the failure to properly adjust pickers at catching.

Inadequate stunning (voltage and time) can lead to petechial hemorrhages usually occurring in the breast and legs. The presence of pathogens in the farm, such as IBDV (Gumboro disease), may increase capillary weakness which leads to carcass bruising.

Mycotoxins such as aflatoxins work in a similar way by reducing the force required to produce bruises due to increased capillary fragility. Usually these occur in the thighs. For mycotoxin-related problems, prevention can be undertaken through the use of a proper mycotoxin risk management tool which relies on complementary detoxification strategies (biotransformation, adsorption, bioprotection) can eliminate the toxic effects in the animals, while guaranteeing liver and immune protection.



Checklist Corrective action

Potential cause: MANAGEMENT: Stunning system

- Voltage of stunning system
- Duration of electric shock
- Correct voltage and timing of electric shocks

Potential cause: PATHOGENS: Infectious bursal disease (IBDV/Gumboro)

- Blood spots mainly located in the legs and the breast
- Necropsy: Bursa of Fabricius is swollen, enlarged and bloody
- Maternal antibody titres are very low in day-old chicks
- Implement/correct vaccination program in breeders
- Change from mild to strong-reaction vaccine
- Correct vaccination age (Deventer formula)
- Increase biosecurity level

Potential cause: MYCOTOXINS: Aflatoxins

- Detection in raw materials (ELISA) or feed (HPLC)
- Animals present jaundice-like symptoms, are dehydrated and emaciated. They present purple-reddish areas in the carcase
- Raw materials originating from supplier/region with history of aflatoxin contamination
- Histopathology: Check other target organs (for example liver)
- Decrease in overall performance of the flock

- Check average contamination levels.
- Use Mycofix® at a correct dosage level.
- Avoid feed bins or feed/water lines becoming contaminated by stale, wet or moldy feed

Potential cause: MANAGEMENT: Animal density

- High flock density at grow-out house
- Adjust flock density

Potential cause: MANAGEMENT: Catching

- Poor catching procedures
- Adjust picking machines and/or catching procedure

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What's Wrong with My Birds? Part 7: Avian Gout/Kidney Failure



Avian gout is a consequence of kidney damage which can occur from a number of potential causes leading to the accumulation of uric acid/urates in the renal tubules and serous coats of the heart, the liver, the mesentery, the air sacs or the peritoneum.

ue to its complex aetiology, it is difficult to diagnose; however, the most common signs are dehydration, pale combs, depression and swelling and reddening of the feet which impair bird movement. In layers, where it is mainly observed, avian gout can lead to mortalities up to 50%, with 19-35 week-old hens mostly affected.

The causes for this condition are varied (see table right), ranging from management and/or nutrition-related, to pathogens and/or the presence of mycotoxins in feed. In terms of nutrition, special attention must be paid to the calcium/phosphorus balance, sodium and vitamin D₃.

In general, any condition favouring an increase of uric acid in blood favours precipitation in tissue and, as a consequence, development of gout. Excess dietary calcium with low available phosphorus results in the precipitation of sodium-urate crystals and calcium pyrophospate (pseudogout). In younger birds, gout due to sodium intoxication may be observed at sodium levels exceeding 0.4% in water and 0.8% in feed.

Likewise, high levels of vitamin D₃ can increase calcium absorption from the intestine favouring the formation and deposition of urate crystals. Also nutrition-related is the protein level in feed which in excess of 30% causes uric acid production leading to excretory loads in kidneys. Concurrently, sulphates decrease calcium resorption causing excess calcium secretion through the urine. This favors gout, as well as any other factor contributing to urine alkalinity. Water deprivation falls in this category as it leads to increased concentrations of uric acid and other minerals in the blood and later on in the kidneys and urine.

Viruses such as infectious bursal disease (IBDV) and/or infectious bronchitis can enhance mortalities in the presence of preexisting kidney damage.

In terms of mycotoxin contamination of feeds, the nephrotoxic aflatoxins (Afla), ochratoxin A (OTA) and citrinin are of major concern. The impairment of the kidney function which results from the action of these mycotoxins reduces uric acid excretion and results in the accumulation of uric acid in the body.

Potential cause: MYCOTOXINS: Ochratoxin A (OTA), citrinin, aflatoxins (Afla)

- Positive for Afla, citrinin and/or OTA in raw materials (ELISA) or feed (HPLC)
- Raw materials originating from supplier/ region with history of mycotoxin contamination
- Histopathology: Check other target organs of these mycotoxins (e.g. kidneys, liver)
- Decline in overall flock performance
- Check average contamination levels
- Use Mycofix® at the correct dosage level
- Avoid feed bins or feed/water lines becoming contaminated by stale, wet or mouldy feed

Potential cause: NUTRITION: Calcium, sodium, vitamin D.

- Level of minerals and vitamins in diets
- Correct level of minerals and vitamin D,
- Control fish meal usage (rich in salt)
- Control total sodium chloride content in feed (<0.3%)

Potential cause: NUTRITION: Protein

Protein level in feeds

• Correct protein level in feeds

Potential cause: MANAGEMENT: Water deprivation

- Observe animal behavior to understand the cause of water deprivation
- Transportation and vaccination procedures
- Drinkers in terms of number, position and blockages that may impede access.
- Chemicals added to water (disinfectants, copper sulphate, etc) may result in water refusal, dehydration and gout
- Improve transportation condition of birds (access to water)
- Adjust number, position and access to drinkers
- Avoid overcrowding
- Correct blockages in nipples

Potential cause: PATHOGENS: Infectious bursal disease (IBDV/Gumboro)

- Maternal antibody titres are very low in day-old chicks
- Adapt vaccination program to the demands of the field situation in each particular area/epidemiology
- Increase biosecurity level

Potential cause: PATHOGENS: Infectious bronchitis (IB)

- Laboratory tests to confirm the presence of the coronavirus in a swab or tissue sample
- Adapt vaccination program to the demands of the field situation in each particular area/epidemiology

References are available on request

For more information, visit www.mycotoxins.info

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What's Wrong with My Birds? Part 8: Lameness conditions (nutrition)



hickens raised for commercial meat production are selectively bred to reach marketweight quickly. This rapid growth, however, can place increasing demands on the bird'sskeletal system resulting in impaired loco-motion. Lameness and gait abnormalities in poultry are conditions of high significancenot only because of their implications interms of animal welfare, but also due to thefinancial losses caused by increased mortal-ity, reduced feed utilisation and growth rate, and downgrading in the processing plant. Nutrition plays

a significant role in skeletalhealth and development, thus a multitude of nutritional factors can lead to musculo-skeletal diseases, which are com monly characterised by lameness. It is important to identify and understand these risk factors inorder to develop a prevention or mitigationstrategy to reduce incidence of lameness inpoultry flocks. The purpose of this table isto outline several nutritional factors that cancontribute to increased incidence of lameness in poultry and offer approaches tohelp mitigate the damage caused by these conditions.

Condition	Corrective action	
RICKETS Potential cause: Vitamin D3 deficiency, Ca/P imbalance		
 Symptoms: Enlargement of the ends of tibia and femur, with widened epiphyseal plate Lesions: Disorganised cartilage matrix, irregular vascular penetration 	Feed vitamin D3 with balanced calcium and phosphorus	
PEROSIS/CHONDRODYSTROPHY Potential cause: Manganese deficie	ncy	
 Symptoms: Thickened and shortened legs, shortened wings Lesions: Enlargement and malformation of the tibio-metatarsal joint, twisting and bending of tibial distal end and the proximal end of tarso-metatarsus, slippage of the gastrocnemius tendon from its chondyles 	 Feed appropriate manganese as per the production stage Maintain Mn/Ca/P balance 	
OSTEOPOROSIS/CAGE LAYER FATIGUE Potential cause: Impaired calc	ium flux in laying hens	
 Symptoms: Soft and rubbery bones, birds on their sides in the back of the cage Lesions: Vertebral fracture affecting spinal cord 	 Feed appropriate calcium as per the production stage Care must be taken to feed ~50% of the dietary calcium in the form of coarse limestone, with the remaining half as fine particle limestone 	
IONOPHORE TOXICITY Potential cause: Monensin		
Symptoms: Legs extended backwardLesions: No specific lesions	Mix feed properlyWithdraw the ionophore	
PODODERMATITIS/FOOTPAD DERMATITIS/FOOT BURN/AMMONIA BURN Potential cause: Biotin deficiency, poor quality litter		
 Symptoms: Ulceration of the metatarsal and digital footpads Lesions: Necrotic lesions on the plantar surface of the footpads 	 Improve gut integrity by feeding multi-species, poultry-specific, live probiotics Supplement biotin in the feed Lower litter moisture with proper ventilation and avoid water spillage 	
TIBIAL DYSCHONDROPLASIA/OSTEOCHONDROSIS Potential cause: Ca/P ratio, excess chloride in feed metabolic acidosis, acid/base balance, mycotoxins		
 Symptoms: Swelling and bowing in the region of the knee joints, angulations of legs, typically in birds >35 days Lesions: Plug of cartilage in proximal end of tibia, distal tibia, and proximal metatarsus, in decreasing order of frequency 	Correct the nutritional imbalancesAdd an effective mycotoxin deactivator in the feed	

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What's Wrong with My Birds? Part 9: Lameness conditions (bacterial pathogens)



n 50 years, broiler growth rates have increased dramatically due to intense genetic selection and enhanced nutritional programs. Fast growth places great demands on birds' musculoskeletal systems, which can result in impaired locomotion and lameness.

Lameness reduces animal well-being and has severe economic consequences from poor growth, increased culling and mortality, and increased carcase condemnation and downgrading at slaughter.

Lameness is often a multifactorial condition. Understanding the various causes can help producers identify areas for improvement and develop effective strategies to reduce the incidence of lameness in their flocks. Conditions responsible for lameness can be of infectious or non-infectious origin. This table focuses on lameness conditions caused by bacterial pathogens and suggests solutions that can help prevent or alleviate lameness caused by these conditions.

Condition	Corrective action
Bacterial Chondronecrosis with Osteomyelitis (BCO)	
 Etiology: Enterococcus cecorum, Streptococcus spp., Staphylococcus spp, E. coli Symptoms: Bird will be sitting on its breast/keel, with the legs directed forward, use of wings for walking support and hip flexion Lesions: Necrotic degeneration and microbial infection, primarily within the proximal heads of the femur and tibia 	 Prevention: Improve gut integrity by feeding multi-strain poultry-specific live probiotic Treatment: Antibiotics depending on severity, but mostly birds are euthanised
Vertebral Osteomyelitis/Spondylitis/Spondylopathy/Spondylolisthesis/Kinkyback	
 Etiology: Enterococcus cecorum, Staphylococcus spp., E. coli Symptoms: Typically starts from day 22, bird will be sitting on its breast/keel, with the legs directed forward, posterior paralysis due to spinal cord compression Lesions: Abscess and/or necrosis in T4-T7 vertebrae, dorsal buckling of spinal cord (kyphosis), interstitial oedema, atrophy, degeneration of muscle fibres 	 Prevention: Improve gut integrity by feeding multi-strain poultry-specific live probiotic Treatment: Antibiotics depending on severity, but mostly birds are euthanised
Bumble Foot	
 Etiology: Staphylococcus spp. Symptoms: Swelling above the hock and around the hocks and feet. Lesions: Abscess on hock joint, infected joints may have clear exudate with fibrin clots 	 Prevention: Improve gut integrity by feeding multi-strain poultry-specific live probiotic Treatment: Antibiotics
Fowl Cholera	
 Etiology: Pasteurella multocida Symptoms: Swollen hock joints, swollen wattles and comb, greenish diarrhoea Lesions: Necrotic foci on liver, petechiae in the epicardial fatty tissues 	Prevention: Vaccines only if endemicTreatment: Antibiotics
Osteomyelitis Complex	
 Etiology: Bacterial, but no specific pathogen has been identified Symptoms: None Lesions: Green discolouration of liver, inflammatory lesions in bones and joints 	 Prevention: Improve gut integrity by feeding multi-strain poultry-specific live probiotic Only identified at slaughter
Bacterial arthritis/Airsacculitis (MS)	
 Etiology: Enterococcus faecalis, Mycoplasma synoviae Symptoms: Ruffled feathers, swollen hock joints and feet, bilaterally asymmetrical legs Lesions: Joints and tendon sheaths have viscous grey to yellow exudate, caseous exudate from the lesions 	 Prevention: Improve gut integrity by feeding multi-strain poultry-specific live probiotic Treatment: Antibiotics and eradication of infected breeding stock

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What's Wrong with My Birds? Part 10: Lameness conditions (viral pathogens)



n 50 years, broiler growth rates have increased dramatically due to intense genetic selection and enhanced nutritional programs. Fast growth places great demands on birds' musculoskeletal systems, which can result in impaired locomotion and lameness. Lameness reduces animal wellbeing and has severe economic consequences from poor growth, increased culling and mortality, increased carcase condemnation and downgrading at slaughter. Lameness is often a multifactorial condition.

Understanding various causes can help producers identify improvements and develop effective strategies to reduce the incidence of lameness in their flocks. Conditions responsible for lameness can be of infectious or non-infectious origin.

This table focuses on lameness conditions caused by pathogenic factors, namely viruses, and suggests solutions that can help prevent or alleviate lameness caused by these conditions.

Condition	Corrective action
Tenosynovitis/Viral arthritis	
 Aetiology: Avian reovirus Symptoms: Soft swelling of the joints with turbid fluid in the capsule, swollen shanks Lesions: Swelling and petechiae in the synovial membranes, small erosions on the articular cartilage, adhesions between the tendons and fibrosis of tissues 	 Prevention: Live vaccine followed by inactivated vaccine Treatment: Euthanise the infected flock
Amyloidosis	
 Symptoms: Swollen hock joint containing orange-yellowish material, muscular shivering Lesions: Extracellular build up of amyloid protein in joints and internal organs 	 Prevention: Live vaccine Treatment: Sodium salicylate 1g/litre (acute phase). Antibiotics to control secondary colibacillosis
Infectious bronchitis (IB)/Infectious laryngo tracheitis (ILT)	
 Aetiology: Corona virus, Herpes virus Symptoms: Sudden death, muscular shivering Lesions: Oedema of skeletal and pectoral muscles 	 Prevention: Live vaccine Treatment: Sodium salicylate 1g/litre (acute phase). Antibiotics to control secondary colibacillosis
Marek's disease	
 Aetiology: Avian Herpes virus 2 Symptoms: One leg stretched forward and the other backward Lesions: Tumors in internal organs, unilateral enlargement of peripheral nerves 	Prevention: Live vaccine Treatment: Eradication of infected flock
Avian encephalomyelitis (AE)	
 Aetiology: Picornavirus Symptoms: Trembling of the head, neck, and wings, paralysis of both legs extended out to one side Lesions: Gross lesions are mild or absent, focal white areas in gizzard muscle 	Prevention: Vaccination of breedersTreatment: None
Newcastle disease	
 Aetiology: Avian paramyxovirus serotype 1 Symptoms: Twisting of neck and paralysis of wings and legs, cyanosis of comb, facial oedema, green diarrhoea, drop in egg production, sudden death Lesions: Haemorrhage in intestine, petechial haemorrhage in proventriculus, congestion and mucoid exudates seen in the respiratory tract, especially in trachea 	 Prevention: Live vaccine Treatment: None. Antibiotics to control secondary bacterial infections
Eastern equine encephalitis	
 Aetiology: Arbovirus Symptoms: Flaccid neck, staggering, paralysis Lesions: No gross lesions 	 Prevention: Vaccination. Control mosquito population Treatment: None

For more information, visit www.mycotoxins.info

What's Wrong with My Birds? Part 11: Lameness conditions (management)



ue to intense genetic selection for increased growth and feed efficiency, lameness has become a growing issue in today's broiler industry on a global scale. Lameness is not only a concern in terms of

animal welfare: it also poses a serious financial threat to poultry producers as it is a significant cause of culling, mortality, and condemnations. Overall, the economic cost associated with lameness problems in poultry can add up to several hundred million dollars each year.

Many risk factors, including both nonpathogenic and pathogenic

causes, could be associated with the occurrence of lameness in broilers and the condition is usually multifactorial.

It is important to differentiate the multiple causes of lameness in order to develop proper prevention and treatment strategies as these strategies will change based on the causative agent identified.

Management factors, such as litter quality and stocking density, can play a major role in the development of leg issues and lameness.

This table highlights several management factors that are commonly associated with increased occurrence of lameness and offers solutions to help mitigate the consequences of these conditions.

Condition	Corrective action
Pododermatitis/Footpad dermatitis/Foot burn/Ammonia burn	
 Causation: Poor litter quality, biotin deficiency Symptoms: Ulceration of the metatarsal and digital footpads Lesions: Necrotic lesions on the plantar surface of the footpads 	 Lower litter moisture with proper ventilation and avoid water spillage Improve gut integrity by feeding poultry- specific, live probiotics Supplement biotin in the feed
Tibial dyschondroplasia/Osteochondrosis	
 Causation: Genetic selection, late rapid growth rate, calcium-phosphorus ratio, excess chloride in feed metabolic acidosis, acid/base balance, mycotoxins Symptoms: Swelling and bowing in the region of the knee joints, angulations of legs typically in birds >35 days Lesions: Plug of cartilage in proximal end of tibia, distal tibia, and proximal metatarsus, in decreasing order of frequency 	 Lower the energy and protein density of feed to slow down the growth Correct the nutritional imbalances Add an effective mycotoxin deactivator in the feed
Twisted leg	
 Causation: Genetic selection, stocking density Symptoms: Distortion at hock, valgus/varus, various angulations of legs Lesions: Linear twisting of tibia and femur, Changed angulation of tibial condyles 	Euthanise affected bird
Degenerative joint disease	
 Causation: Developmental defects, physical damage Symptoms: Imbalanced walking, huddling Lesions: Damaged epiphyseal articular cartilage, especially of femoral anti-trochanter, but also other leg joints, resulting in erosions and cartilage flaps 	Euthanise affected bird
Ionophore toxicity	
 Causation: Monensin Symptoms: Legs extended backward Lesions: No specific lesions 	 Mix feed properly Withdraw the ionophore

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